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\* \* \* \* \* STN Columbus \* \* \* \* \*

FILE 'HOME' ENTERED AT 19:07:44 ON 18 OCT 2007

=> file medline embase biosis scisearch caplus  
'SCISEARCH' IS NOT A VALID FILE NAME

Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files that are available. If you have requested multiple files, you can specify a corrected file name or you can enter "IGNORE" to continue accessing the remaining file names entered.

ENTER A FILE NAME OR (IGNORE):scisearch

'SCISEARCH' IS NOT A VALID FILE NAME

Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files that are available. If you have requested multiple files, you can specify a corrected file name or you can enter "IGNORE" to continue accessing the remaining file names entered.

ENTER A FILE NAME OR (IGNORE):ignore

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
0.21	0.21

FULL ESTIMATED COST

FILE 'MEDLINE' ENTERED AT 19:08:31 ON 18 OCT 2007

FILE 'EMBASE' ENTERED AT 19:08:31 ON 18 OCT 2007

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FILE 'BIOSIS' ENTERED AT 19:08:31 ON 18 OCT 2007

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=> file scisearch

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
3.26	3.47

FULL ESTIMATED COST

FILE 'SCISEARCH' ENTERED AT 19:08:51 ON 18 OCT 2007

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FILE COVERS 1974 TO 11 Oct 2007 (20071011/ED)

SCISEARCH has been reloaded, see HELP RLOAD for details.

=>

---Logging off of STN---

=>

Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
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FULL ESTIMATED COST

ENTRY      SESSION  
2.61      6.08

STN INTERNATIONAL LOGOFF AT 19:09:15 ON 18 OCT 2007

Connecting via Winsock to STN

Welcome to STN International! Enter x:X

LOGINID:SSSPTA1644PNH

PASSWORD:

TERMINAL (ENTER 1, 2, 3, OR ?):2

\* \* \* \* \* Welcome to STN International \* \* \* \* \*

NEWS	1		Web Page for STN Seminar Schedule - N. America
NEWS	2	JUL 02	LMEDLINE coverage updated
NEWS	3	JUL 02	SCISEARCH enhanced with complete author names
NEWS	4	JUL 02	CHEMCATS accession numbers revised
NEWS	5	JUL 02	CA/CAPplus enhanced with utility model patents from China
NEWS	6	JUL 16	CAPplus enhanced with French and German abstracts
NEWS	7	JUL 18	CA/CAPplus patent coverage enhanced
NEWS	8	JUL 26	USPATFULL/USPAT2 enhanced with IPC reclassification
NEWS	9	JUL 30	USGENE now available on STN
NEWS	10	AUG 06	CAS REGISTRY enhanced with new experimental property tags
NEWS	11	AUG 06	BEILSTEIN updated with new compounds
NEWS	12	AUG 06	FSTA enhanced with new thesaurus edition
NEWS	13	AUG 13	CA/CAPplus enhanced with additional kind codes for granted patents
NEWS	14	AUG 20	CA/CAPplus enhanced with CAS indexing in pre-1907 records
NEWS	15	AUG 27	Full-text patent databases enhanced with predefined patent family display formats from INPADOCDB
NEWS	16	AUG 27	USPATOLD now available on STN
NEWS	17	AUG 28	CAS REGISTRY enhanced with additional experimental spectral property data
NEWS	18	SEP 07	STN AnaVist, Version 2.0, now available with Derwent World Patents Index
NEWS	19	SEP 13	FORIS renamed to SOFIS
NEWS	20	SEP 13	INPADOCDB enhanced with monthly SDI frequency
NEWS	21	SEP 17	CA/CAPplus enhanced with printed CA page images from 1967-1998
NEWS	22	SEP 17	CAPplus coverage extended to include traditional medicine patents
NEWS	23	SEP 24	EMBASE, EMBAL, and LEMBASE reloaded with enhancements
NEWS	24	OCT 02	CA/CAPplus enhanced with pre-1907 records from Chemisches Zentralblatt
NEWS EXPRESS	19	SEPTEMBER 2007: CURRENT WINDOWS VERSION IS V8.2, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 19 SEPTEMBER 2007.	
NEWS HOURS	STN Operating Hours Plus Help Desk Availability		
NEWS LOGIN	Welcome Banner and News Items		
NEWS IPC8	For general information regarding STN implementation of IPC 8		

Enter NEWS followed by the item number or name to see news on that specific topic.

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FILE 'HOME' ENTERED AT 19:09:34 ON 18 OCT 2007

=> file medline embase biosis scisearch caplus  
COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
0.21	0.21

FULL ESTIMATED COST

FILE 'MEDLINE' ENTERED AT 19:09:49 ON 18 OCT 2007

FILE 'EMBASE' ENTERED AT 19:09:49 ON 18 OCT 2007  
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=> s KDR  
L1 9883 KDR

=> s l1 or Flk-1 or VEGFR-2  
L2 18983 L1 OR FLK-1 OR VEGFR-2

=> s l2 and peptide  
L3 1160 L2 AND PEPTIDE

=> s l3 and HLA  
L4 26 L3 AND HLA

=> dup remove l4  
PROCESSING COMPLETED FOR L4  
L5 22 DUP REMOVE L4 (4 DUPLICATES REMOVED)

=> s l5 and pd<20030307  
1 FILES SEARCHED...  
4 FILES SEARCHED...  
L6 4 L5 AND PD<20030307

=> d l6 1-4 cbib abs

L6 ANSWER 1 OF 4 MEDLINE on STN  
2001321766. PubMed ID: 11211940. Dendritic cells derived from peripheral monocytes express endothelial markers and in the presence of angiogenic growth factors differentiate into endothelial-like cells. Fernandez Pujol B; Lucibello F C; Zuzarte M; Lutjens P; Muller R; Havemann K. (Institute of Molecular Biology and Tumor Research, University of Marburg, Germany. ) European journal of cell biology, (2001 Jan) Vol. 80, No. 1, pp. 99-110. Journal code: 7906240. ISSN: 0171-9335. Pub. country: Germany: Germany, Federal Republic of. Language: English.  
AB CD14-positive monocytes obtained from human peripheral blood were cultured

with GM-CSF and IL-4. During the early culture phase immature dendritic cells (DCs) developed which not only expressed CD1a, **HLA-DR** and CD86, but also expressed the endothelial cell markers von Willebrand factor (vWF), VE-cadherin and VEGF receptors Flt-1 and Flt-4. Further maturation of DCs was achieved by prolonged cultivation with TNF $\alpha$ . These cells showed typical DC morphology and like professional antigen-presenting cells (APCs) expressed CD83 and high levels of **HLA-DR** and CD86. However, if immature DCs were grown with VEGF, bFGF and IGF-1 on fibronectin/vitronectin-coated culture dishes, a marked change in morphology into caudated or oval cells occurred. In the presence of these angiogenic growth factors the cultured cells developed into endothelial-like cells (ELCs), characterized by increased expression of vWF, **KDR** and Flt-4 and a disappearance of CD1a and CD83. Addition of IL-4 and Oncostatin M also increased VE-cadherin expression, and the loosely adherent cells formed clusters, cobblestones and network-like structures. vWF- expressing ELCs mainly originated from CD1a-positive cells, and VEGF was responsible for the decrease in the expression of the DC markers CD1a and CD83. In mixed leukocyte cultures, mature DCs were more potent APCs than ELCs. Moreover, Ac-LDL uptake, and the formation of tubular structures on a plasma matrix was restricted to ELCs. These results suggest that in the presence of specific cytokines immature DCs have the potential to differentiate along different lineages, i.e. into a cell type resembling ELCs.

L6 ANSWER 2 OF 4 MEDLINE on STN  
1999300696. PubMed ID: 10372108. Expression of novel surface antigens on early hematopoietic cells. Buhring H J; Seiffert M; Bock T A; Scheduling S; Thiel A; Scheffold A; Kanz L; Brugger W. (Department of Hematology and Oncology, University of Tübingen, Germany.. hjbuehri@med.uni-tuebingen.de) . Annals of the New York Academy of Sciences, (1999 Apr 30) Vol. 872, pp. 25-38; discussion 38-9. Journal code: 7506858. ISSN: 0077-8923. Pub. country: United States. Language: English.

AB The purpose of this report is to demonstrate the expression of very recently identified surface antigens on CD34+ and AC133+ bone marrow (BM) cells. Coexpression analysis of AC133 and defined antigens on CD34+ BM cells revealed that the majority of the CD164+, CD135+, CD117+, CD38low, CD33+, and CD71low cells resides in the AC133+ population. In contrast, most of the CD10+ and CD19+ B cell progenitors and a fraction of the CD71high population are AC133-, indicating that CD34+AC133+ cells are enriched in primitive and myeloid progenitor cells, whereas CD34+AC133- cells mainly consist of B cell and late erythroid progenitors. This corresponds to the highly reduced percentage of CD10+ B cells and the absence of CD71high erythroid progenitors on AC133+ selected BM cells. A portion of 0.2-0.7% of the AC133+ selected cells do not coexpress CD34. These cells are very small and define a uniform CD71-, CD117-, CD10-, CD38low, CD135+, **HLA-DR**high, CD45+ population with unknown delineation. Four color analysis on CD34+CD38- BM cells revealed that virtually all of these primitive cells express AC133. Using an improved liposome-enhanced labeling technique for the staining of weakly expressed antigens, subsets of this population could be identified which express the angiopoietin receptors TIE (67.6%) and TEK (36.8%), the vascular endothelial growth factor receptors FLT1 (7%), FLT4 (3.2%), and **KDR** (10.4%), or the receptor tyrosine kinases HER-2 (15.4%) and FLT3 (CD135; 77.6%). Our results suggest that the CD34+CD38- population is heterogeneous with respect to the expression of the analyzed receptor tyrosine kinases.

L6 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN  
2007:621423 Document No. 147:16719 Progenitor endothelial cell capturing with a drug eluting implantable medical device. Cottone, Robert John, Jr.; Rowland, Stephen M.; Parker, Sherry (Orbusneich Medical, Inc., USA). U.S. Pat. Appl. Publ. US 2007128723 A1 20070607, 47 pp., Cont.-in-part of U.S. Ser. No. 76,731. (English). CODEN: USXXCO. APPLICATION: US 2006-560352 20061115. PRIORITY: US 2005-76731 20050310; US 2003-442669 20030520; US 2003-360567 20030206; US 2001-808867 20010315; US

2006-822451P 20060815; US 2005-736920P 20051115; US 2002-382095P 20020520;  
US 2002-354680P 20020206; US 2000-201789P 20000504; US 2000-189674P  
20000315.

AB A medical device for implantation into vessels or luminal structures within the body is provided, which stimulates pos. blood vessel remodeling. The medical device, such as a stent and a synthetic graft, is coated with a pharmaceutical composition consisting of a controlled-release matrix and one or more pharmaceutical substances for direct delivery of drugs to surrounding tissues. The coating on the medical device further comprises a ligand such as a **peptide**, an antibody or a small mol. for capturing progenitor endothelial cells in the blood contacting surface of the device for restoring an endothelium at the site of injury. In particular, the drug-coated stents are for use, for example, in balloon angioplasty procedures for preventing or inhibiting restenosis.

L6 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN

2003:971783 Document No. 140:19916 Medical device with coating that promotes cell adherence and differentiation. Kutryk, Michael J. B.; Cottone, Robert J., Jr.; Rowland, Stephen M.; Kuliszewski, Michael A. (Can.). U.S. Pat. Appl. Publ. US 2003229393 A1 20031211, 41 pp., Cont.-in-part of U.S. Ser. No. 808,867. (English). CODEN: USXXCO. APPLICATION: US 2003-360567 20030206. PRIORITY: US 2001-808867 20010315; US 2002-354680P 20020206.

AB Compns. and methods are provided for producing a medical device such as a stent, a stent graft, a synthetic vascular graft, heart valves, coated with a biocompatible matrix which incorporates antibodies, antibody fragments, or small mols., which recognize, bind to and/or interact with a progenitor cell surface antigen to immobilize the cells at the surface of the device. The coating on the device can also contain a compound or growth factor for promoting the progenitor endothelial cell to accelerate adherence, growth and differentiation of the bound cells into mature and functional endothelial cells on the surface of the device to prevent intimal hyperplasia. Methods for preparing such medical devices, compns., and methods for treating a mammal with vascular disease such as restenosis, atherosclerosis or other types of vessel obstructions are disclosed. CMDX-coated stainless steel samples having anti-CD34 antibody bound on its surface. The samples contained numerous adherent cell near confluent monolayer.

=> s "VYSSEEAEL"

L7 0 "VYSSEEAEL"

=> s l1 and GYRIYDVVL

L8 0 L1 AND GYRIYDVVL

=> d his

(FILE 'HOME' ENTERED AT 19:09:34 ON 18 OCT 2007)

FILE 'MEDLINE, EMBASE, BIOSIS, SCISEARCH, CAPLUS' ENTERED AT 19:09:49 ON  
18 OCT 2007

L1 9883 S KDR

L2 18983 S L1 OR FLK-1 OR VEGFR-2

L3 1160 S L2 AND PEPTIDE

L4 26 S L3 AND HLA

L5 22 DUP REMOVE L4 (4 DUPLICATES REMOVED)

L6 4 S L5 AND PD<20030307

L7 0 S "VYSSEEAEL"

L8 0 S L1 AND GYRIYDVVL

=> s l3 and "SYMISYAGM"

L9 0 L3 AND "SYMISYAGM"

=> s l3 and "RFVPDGNRI"

L10 0 L3 AND "RFVPDGNRI"

=> s "KWEFPRDRL"  
L11 0 "KWEFPRDRL"

=> s "DFLTLEHLI"  
L12 0 "DFLTLEHLI"

=> s "AMFFWLLLV"  
L13 0 "AMFFWLLLV"

=> s "VIAMFFWLL"  
L14 0 "VIAMFFWLL"

=> s "AVIAMFFWL"  
L15 0 "AVIAMFFWL"

=> s "KLIEIGVQT"  
L16 0 "KLIEIGVQT"

=> s "YMISYAGMV"  
L17 0 "YMISYAGMV"

=> s "IQSDVWSFGV"  
L18 0 "IQSDVWSFGV"

=> s human vascular growth factor receptor 2  
L19 0 HUMAN VASCULAR GROWTH FACTOR RECEPTOR 2

=> s "human VEGFR2"  
L20 55 "HUMAN VEGFR2"

=> s l20 and CTL epitope  
L21 0 L20 AND CTL EPITOPE

=> s l20 and HLA  
L22 0 L20 AND HLA

=> s l20 and peptide  
L23 2 L20 AND PEPTIDE

=> dup remove l23  
PROCESSING COMPLETED FOR L23  
L24 2 DUP REMOVE L23 (0 DUPLICATES REMOVED)

=> d l24 1-2 cbib abs

L24 ANSWER 1 OF 2 MEDLINE on STN  
2004284100. PubMed ID: 15183093. Human/mouse cross-reactive anti-VEGF  
receptor 2 recombinant antibodies selected from an immune b9 allotype  
rabbit antibody library. Popkov Mikhail; Jendreyko Nina; Gonzalez-Sapienza  
Gualberto; Mage Rose G; Rader Christoph; Barbas Carlos F 3rd. (Department  
of Molecular Biology, The Scripps Research Institute, La Jolla, CA 92037,  
USA. ) Journal of immunological methods, (2004 May) Vol. 288, No. 1-2, pp.  
149-64. Journal code: 1305440. ISSN: 0022-1759. Pub. country:  
Netherlands. Language: English.

AB Vascular endothelial growth factor (VEGF) and its receptors have been  
implicated in promoting solid tumor growth and metastasis via stimulating  
tumor-associated angiogenesis. Models of murine tumor angiogenesis and  
receptor-specific antibodies are required to evaluate roles of VEGF  
receptors in mouse models of human cancer. **Human VEGFR2**  
(also known as KDR) and murine VEGFR2 (or Flk-1) share 85% amino acid  
sequence identity in their extracellular domain. We describe here the  
development of antibodies that cross-react with mouse and **human**  
**VEGFR2**. High-affinity, species cross-reactive, Fabs specific for  
KDR/Flk-1 were selected from an antibody phage display library generated

from an immunized rabbit of b9 allotype. The selected chimeric rabbit/human Fabs were found to bind to purified KDR and Flk-1 with nanomolar affinity. Three of the selected Fabs detected KDR expression on human endothelial cells as well as Flk-1 on murine endothelial cells. The availability of anti-VEGFR2 Fab with species cross-reactivity will help to decipher the functional role of KDR/Flk-1 in tumor biology as well as facilitate the preclinical evaluation of the suitability of KDR/Flk-1 for drug targeting. This report underscores our earlier finding that b9 rabbits are excellent sources for high-affinity cross-reactive antibodies with therapeutic potential.  
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L24 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN

2003:850821 Document No. 140:92257 Tailoring in vitro selection for a picomolar affinity human antibody directed against vascular endothelial growth factor receptor 2 for enhanced neutralizing activity. Lu, Dan; Shen, Juqun; Vil, Marie D.; Zhang, Haifan; Jimenez, Xenia; Bohlen, Peter; Witte, Larry; Zhu, Zhenping (Department of Antibody Technology, ImClone Systems Incorporated, New York, NY, 10014, USA). Journal of Biological Chemistry, 278(44), 43496-43507 (English) 2003. CODEN: JBCHA3. ISSN: 0021-9258. Publisher: American Society for Biochemistry and Molecular Biology.

AB Vascular endothelial growth factor (VEGF) and its receptors have been implicated in promoting solid tumor growth and metastasis via stimulating tumor-associated angiogenesis. We previously identified several fully human neutralizing anti-VEGF receptor 2 (or kinase inserting domain-containing receptor (KDR)) antibodies from a large antibody phage display library. These antibodies bind specifically to KDR, block VEGF/KDR interaction, and inhibit VEGF-induced proliferation of human endothelial cells and migration of KDR+ leukemia cells. Three of these antibodies, interestingly, share an identical heavy chain variable (VH) sequence. In this report, we constructed a new library comprising the single VH paired with the variable light chain (VL) repertoire obtained from the original naive human library. Initial in vitro selection revealed that the single VH could pair with a number of different VL while retaining its specificity for KDR. However, a consensus VH/VL pair, clone 1121, was identified after three or four rounds of selection by tailoring the stringency of the panning conditions. Clone 1121 showed a >30-fold higher binding affinity to KDR (Kd, 100 pM) because of improvement on both association and dissociation consts. and blocked VEGF/KDR interaction with an IC50 of .apprx.1 nM, compared with that of 3-4 nM for the parent Fab fragments. Further, clone 1121 was more potent in inhibiting VEGF-stimulated KDR phosphorylation in endothelial cells. A binding epitope mapping study on clone 1121 and one of the parent clones, 2C6, demonstrated that both antibodies interacted with the third Ig domain within the extracellular region of KDR. Several **peptide** phage display libraries were utilized to further examine the fine binding specificities of the two antibodies. All of the 2C6-binding **peptides** are cysteine-constrained, whereas clone 1121 binds to both cysteine-constrained and linear **peptides**. It is noteworthy that most of the 2C6-binding **peptides** also cross-react with clone 1121, but none of the clone 1121-specific **peptides** binds to 2C6, indicating that clone 1121 retained part of the original binding epitope(s) of 2C6 while gaining new binding specificity. Taken together, our observation suggests that clone 1121 may have great clin. potential in anti-angiogenesis therapy. It further underscores the efforts to identify antibodies of high affinity for enhanced biol. activities.

=> s CTL epitope

L25 6703 CTL EPITOPE

=> s l25 and KDR

L26 1 L25 AND KDR

=> d 126 cbib abs

L26 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2007 ACS on STN

2000:240985 Document No. 132:292701 Novel methods for therapeutic vaccination. Steinaa, Lucilla; Mouritsen, Soren; Nielsen, Klaus Gregorious; Haaning, Jesper; Leach, Dana; Dalum, Iben; Gautam, Anand; Birk, Peter; Karlsson, Gunilla (M & E Biotech A/S, Den.). PCT Int. Appl. WO 2000020027 A2 20000413, 220 pp. DESIGNATED STATES: W: AE, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1999-DK525 19991005. PRIORITY: DK 1998-1261 19981005; US 1998-PV105011 19981020.

AB A method is disclosed for inducing cell-mediated immunity against cellular antigens. More specifically, the invention provides for a method for inducing cytotoxic T-lymphocyte immunity against weak antigens, notably self-proteins. The method entails that antigen presenting cells are induced to present at least one CTL epitope of the weak antigen and at the same time presenting at least one foreign T-helper lymphocyte epitope. In a preferred embodiment, the antigen is a cancer specific antigen, e.g. prostate specific membrane antigen (PSM), Her2, or FGF8b. The method can be exercised by using traditional polypeptide vaccination, but also by using live attenuated vaccines or nucleic acid vaccination. The invention furthermore provides immunogenic analogs of PSM, Her2 and FGF8b, as well as nucleic acid mols. encoding these analogs. Also vectors and transformed cells are disclosed. The invention also provides for a method for identification of immunogenic analogs of weak or non-immunogenic antigens.

=> s 125 and VEGFR2

L27 0 L25 AND VEGFR2

=> s 125 and FLK-1

L28 0 L25 AND FLK-1

=>

---Logging off of STN---

=>

Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

100.49

100.70

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE

TOTAL

ENTRY

SESSION

CA SUBSCRIBER PRICE

-3.12

-3.12

STN INTERNATIONAL LOGOFF AT 19:16:31 ON 18 OCT 2007